ELSEVIER

Contents lists available at ScienceDirect

Journal of Forensic and Legal Medicine

journal homepage: www.elsevier.com/locate/jflm



Case report

Optic nerve trauma with unilateral edema as a result of head injury

Henry J. Carson M.D Fellow ^{a,*}, Daniel Lingamfelter D.O Deputy Medical Examiner ^a, Mary H. Dudley M.D Chief Medical Examiner and Associate Clinical Professor ^{a,b}

^a Office of the Jackson County Medical Examiner, 660 E 24th Street, Kansas City MO 64108, USA

ARTICLE INFO

Article history: Received 13 December 2009 Received in revised form 9 February 2010 Accepted 1 April 2010 Available online 28 April 2010

Keywords:
Optic nerve
Edema
Skull fracture
Blunt force injury

ABSTRACT

We encountered a patient who suffered a head injury that translated to the optic nerves, leading to dramatic unilateral right optic nerve edema. The decedent was a 20-year-old unsecured passenger in a convertible. The car collided with a pickup truck. The patient survived for 8 h. At autopsy, a comminuted skull fracture involving the right frontal bone including the right orbital plate was found. The right optic nerve measured 1.2 cm in diameter, compared to only 0.4 cm for the left optic nerve. Microscopically, the right optic nerve was markedly edematous, but the nerve fibers and nuclei were viable and intact. The dramatic difference in size between the right and left optic nerves can be attributed to several mechanisms. The survival interval was essential for the asymmetrical swelling to take place. Optic nerve trauma is relatively rare in head injuries, reported to be present in 0.5% of automobile accidents and assaults. Other causes of optic nerve trauma and edema include tumors, osteopetrosis, or reaction to a peripheral hemodialysis shunt.

© 2010 Elsevier Ltd and Faculty of Forensic and Legal Medicine. All rights reserved.

1. Introduction

Optic nerve trauma and edema can develop from automobile accidents, ^{1,2} assaults, ¹ tumors, ^{3,4} or exotic causes such as osteopetrosis ⁵ or a peripheral arteriovenous fistula. ⁶ The common feature in these cases is impairment of the venous circulation of the optic nerve, either by direct compression, ^{3,4} venous thrombosis, ⁶ or increased intracranial pressure. ⁵ Some causes of optic nerve edema, particularly those involving trauma, may be multi-factorial.

We recently encountered a patient who suffered a head injury with distinctive direct force to the orbit that translated to the optic nerves, leading to dramatic injury. We report this case to illustrate an unusual pattern of intracranial injury, and to alert examiners to be aware of this unique blunt force injury to the head.

2. Case report

The decedent was a 20-year-old unsecured passenger in a convertible. The driver appeared to have suffered seizure-like activity and lost consciousness while operating the vehicle. The car crossed the centerline and collided with a pickup truck. The driver survived and was transported to the hospital in critical condition. The subject was extracted from the vehicle and transported to the

emergency department, where the evaluation showed him to have a Glascow coma score 3, incontinence, and poor rectal tone. On physical examination, there was a laceration of the left elbow, a right upper extremity contusion, a right shoulder dislocation, and a left ankle fracture. Computed tomography (CT) scan of the head showed a right skull fracture, intracranial swelling, and a C5 fracture of the cervical spinal cord. The injuries were assessed to be unsurvivable, and the patient was declared brain dead. His family consented to organ and tissue donation. The patient lived for approximately 8 h from the time of the accident to organ collection. After organ and tissue procurement, the body was then transferred to our office for autopsy.

The prominent head injuries included right periorbital ecchymosis and a full-thickness laceration of the right forehead. A comminuted skull fracture involved the right frontal bone including the right orbital plate. The right optic nerve measured 1.2 cm in diameter, compared to only 0.4 cm for the left optic nerve (Fig. 1). Diffuse right frontal subdural and diffuse subarachnoid hemorrhages were present. The brain showed right frontal lacerations and contusions, and contusions of the brain stem. There was a transverse fracture of the C5 vertebral body. Other injuries were consistent with those observed clinically.

Microscopically, the right optic nerve was markedly edematous, but the nerve fibers and nuclei were viable and intact. The left optic nerve showed minimal edema and intraparenchymal hemorrhage.

The cause of death was determined to be blunt force injuries. The manner of death was accident.

^b University of Missouri Kansas City School of Medicine, Kansas City MO, USA

^{*} Corresponding author. Tel.: +1 816 881 6600; fax: +1 816 881 6598. E-mail address: hjcmd@earthlink.net (H.J. Carson).

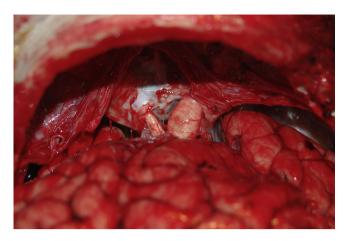


Fig. 1. Optic nerves at autopsy. Note the prominent, swollen right optic nerve compared to the left. A subarachnoid hemorrhage can be appreciated in the frontal lobes, which are reflected away from the skull base.

3. Discussion

The dramatic difference in size and pathological reaction between the right and left optic nerves can be attributed to several mechanisms. The path of the force the automobile accident inflicted on the patient's right orbit caused the primary injury to the right optic nerve, with reactive edema. The skull fracture that involved the right orbital plate, which disrupted but did not transect the optic nerve, further caused it to swell and provided room for it to do so. Common to all of these mechanisms was the fact that the patient survived for at least 8 h after his injury, even though he was brain dead based on physical findings. Had he died immediately at the time of the collision, the impressive difference between the sizes of the optic nerves likely would not have developed.

Optic nerve trauma is relatively rare in head injuries, reported to be present in 0.5% of a British series, predominantly from automobile accidents and assaults. While all deaths in this series had ocular trauma of some sort, over half of the patients with ocular injuries survived with no significant neurologic impairment. Isolated unilateral optic nerve edema has been reported in a patient of a rear-end car accident, in which the patient responded to corticosteroids and recovered.

Obviously, tumors of the optic nerve⁴ or its adjacent structures such as the meninges³ can cause unilateral enlargement and reactive edema of the optic nerve. Recovery depends on the ability to treat the primary or obstructing tumors. Very unusual problems leading to unilateral optic nerve edema have been

reported in osteopetrosis, which can cause solitary nerve compression and reaction,⁵ or in reaction to a peripheral hemodialysis shunt, in which venous thrombosis, stenosis, or hypertension have been identified as possible causes of the reaction in the optic nerve.⁶

In cases of orbital fracture with survival, optic nerve trauma and swelling can lead to visual impairment and blindness.⁷ Surgical decompression of the orbit can allow the nerve to recover and vision to be restored, although the decision to operate must be made on an individual basis, since operative risk may outweigh benefit.^{7,8} Spontaneous recovery of optic nerve function is also possible.⁹ The value of steroids in treating optic nerve swelling may be in dispute, particularly more than 8 h after the initial injury.⁹ Significant visual recovery is possible in spite of serious injury, however, so prudent observation and intervention can be important for managing survivors with this complication.^{7,10}

This young man survived for 8 h in a vegetative state, long enough to develop marked unilateral optic nerve edema. In somebody who died suddenly, such a gross finding likely would not have developed. It is worth being aware of this condition as another sign of prolonged survival, and to note it as a finding.

Conflict of Interest

None declared.

References

- Kulkarni AR, Aggarwal SP, Kulkarni RR, Deshpande MD, Walimbe PB, Labhsetwar AS. Ocular manifestations of head injury: a clinical study. *Eye* 2005; 19:1257–63.
- Maegele M. Reversal of isolated unilateral optic nerve edema with concomitant visual impairment following blunt trauma: a case report. J Med Case Reports 2008;2:50.
- Acebes X, Arruga J, Acebes JJ, Majos C, Muños S, Valero IA. Intracranial meningiomatosis causing Foster Kennedy syndrome by unilateral optic nerve compression and blockage of the superior sagittal sinus. J Neuroophthalmol 2009;29:140–2.
- Eggers H, Jakobiec FA, Jones IS. Tumors of the optic nerve. Doc Ophthalmol 1976;41:43–128.
- Allen RC, Nerad JA, Kattah JC, Lee AG. Resolution of optic nerve edema and improved visual function after optic nerve sheath fenestration in a patient with osteopetrosis. Am J Ophthalmol 2006; 141:945

 –7.
- Chang S, Masaryk TJ, Lee MS. Optic nerve edema: complication of peripheral hemodialysis shunt. Semin Ophthalmol 2004;19:88–90.
- Rinna C, Rocchi G, Ventucci E, Pagnoni M, Iannetti G. Bilateral orbital roof fracture. J Craniofac Surg 2009;20:737–42.
- 8. Yu Wai Man P, Griffiths PG. Surgery for traumatic optic neuropathy. *Cochrane Database Syst Rev*; 2005 October 19. CD005024.
- 9. Yu Wai Man P, Griffiths PG. Steroids for traumatic optic neuropathy. *Cochrane Database Syst Rev*; 2007 October 17. CD006032.
- Lipkin AF, Woodson GE, Miller RH. Visual loss due to orbital fracture. The role of early reduction. Arch Otolaryngol Head Neck Surg 1987;113:81–3.